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From: Gambel, Phillip
Sent: Friday, August 30, 2002 1:52 PM
To: STIC-ILL
Subject: noelle oophoritis

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please provide the following references to

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2/7/5 (Item 5 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

09623681 BIOSIS NO.: 199598078599
Mechanism of self-tolerance and events leading to autoimmune disease and autoantibody response.
AUTHOR: Tung Kenneth S K
AUTHOR ADDRESS: Dep. Pathol., Box 214, Univ. Va., Charlottesville, VA 22908**USA
JOURNAL: Clinical Immunology and Immunopathology 73 (3):p275-282 1994
ISSN: 0090-1229
DOCUMENT TYPE: Literature Review
RECORD TYPE: Abstract
LANGUAGE: English

ABSTRACT: This review summarizes recent studies on the mechanisms of self-tolerance and autoimmune disease pathogenesis based primarily on the murine ovarian autoimmune disease models. The ovarian autoimmune disease was induced experimentally by three approaches: (1) transfer of normal T cells into syngeneic athymic nu/nu mice, (2) neonatal thymectomy, or (3) immunization with a well-defined peptide from the ovarian antigen, ZP3. Self-reactive T cells with capacity to elicit autoimmune oophoritis and autoimmune gastritis are not deleted in the neonatal or adult thymus. In th adult spleen, T cells are not pathogenic until regulatory T cells hav been depleted. Thus the balance of activity between pathogenic T cells and regulatory T cells appears to determine the tolerance status of the host to self-antigens responsible for these autoimmune diseases. Murine autoimmune disease of the ovaries was found to occur through two indep ndent pathways. The first is by depletion of regulatory T cells, as creat d by thymectomy within 4 days after birth. Alternatively, athogenic T cells can be activated through molecular mimicry at the of T cell peptide. This appears to depend on the sharing between for ian and ovarian peptides of critical amino acid residues required recogni of pathogenic T cells. Finally, when T cells that spontaneou peptide are activated, endogenous ovarian antigens can with ZP3 domai ulate B cells to produce antibodies that react ide the immunizing T cell peptide.

2/7/6 (Item 6 from file: 5)
DIALOG(R)File 5:Biosis Previews(

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08026002 BIOSIS NO.: 000093070925
AUTOIMMUNE DISEASE OF THE OVARY INDUCED BY A ZP3 PEPTIDE FROM THE MOUSE ZONE PELLUCIDA
AUTHOR: RHIM S H; MILLAR S E; ROBEY F; LUO A-M; LOU Y-H; YULE T; ALLEN P; DEAN J; TUNG K S K
AUTHOR ADDRESS: DEP. PATHOL., UNIV. VA., CHARLOTTESVILLE, VA. 22908.
JOURNAL: J CLIN INVEST 89 (1). 1992. 28-35. 1992
FULL JOURNAL NAME: Journal of Clinical Investigation
CODEN: JCINA
RECORD TYPE: Abstract
LANGUAGE: ENGLISH

ABSTRACT: We describe a novel experimental system in mice for the study of ovarian autoimmune disease, a condition encountered in women with premature ovarian failure. The ovarian autoimmune disease is induced in B6AF1 mice by a 15-amino acid peptide (Cys-Ser-Asn-Ser-Ser-Ser-Ser-Gln-Phe-Gln-Ile-His-Gly-Pro-Arg) from mouse ZP3, the sperm-binding component of the zona pellucida that surrounds growing and mature oocytes. Whereas the peptide induces both T cell and antibody responses, adoptive transfer of CD4+ T cell lines derived from affected animals causes oophoritis without observable antibodies to the zona pellucida peptide. The primacy of the T cell response in the pathogenesis of disease is further substantiated by defining oophoritogenic peptides as small as eight amino acids (Asn-Ser-Ser-Ser-Ser-Gln-Phe-Gln) that do not elicit an antibody response to the full-length ZP3 peptide. The identification of a well characterized peptide as a causative agent of autoimmune oophoritis should facilitate understanding of the pathogenesis of this T cell-mediated autoimmune disease. Because the proteins of the zona pellucida are conserved among mammals (the mouse and human ZP3 proteins are 67% identical), this murine model may lead to better understanding of the pathogenesis of human autoimmune oophoritis.

L. Phillip Dambd
1644 8/30

2/7/12 (Item 5 from file: 73)
DIALOG(R)File 73:EMBASE

03075364 EMBASE No: 1986232941
Lymphocyte dysfunction in autoimmune oophoritis. Resumption of menses with corticosteroids
Rabinowe S.L.; Berger M.J.; Welch W.R.; Dluhy R.G.
Department of Medicine, Brigham and Women's Hospital, Boston, MA 02115 United States
American Journal of Medicine (AM. J. MED.) (United States) 1986, 81/2 (347-350)
CODEN: AJMEA
DOCUMENT TYPE: Journal
LANGUAGE: ENGLISH

In a 32-year-old woman with secondary amenorrhea and biopsy-proven oophoritis, the circulating T lymphocytes were examined utilizing monoclonal antibody L243 to the nonpolymorphic region of the Ia antigen. The percentage of peripheral T cells expressing the Ia 'immune-associated' antigen was 5.6 percent (normal 3 percent or less). With corticosteroid therapy, the percentage decreased to 2 percent and menses resumed after secondary amenorrhea or two years' duration. Following cessation of steroid administration, the percentage of Ia-positive T cells rose to 7.0 percent and secondary amenorrhea redeveloped in the patient. After corticosteroid therapy was reinstituted, menses resumed and the percentage of Ia-positive T cells fell to normal. This report represents additional new evidence of immune dysfunction in patients with 'autoimmune' oophoritis.